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TWO CASES OF SYRINGO-MYELIA.

BY

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TWO CASES OF SYRINGOMYELIA.[1]

By JOHN AMORY JEFFRIES, M. D.

FEMALE, single, twenty-five years old; sick for ten years; lateral curvature; paronychia; muscular atrophy of right arm, much diminished senses of heat, cold and pain in right arm, neck and side of thorax; spot of same on left thigh; touch perfect; left pupil larger than right.

November 14th, 1889. M. C., with no family history of nervous disease; by nature left-handed; began nine or ten years ago to suffer on sneezing, from a very severe stabbing pain, radiating over the body from about the second dorsal vertebrae. Six months later it became difficult to hold a pen in the right hand, and shortly a "run around" appeared on the thumb. This "was sore, but did not hurt much." After recovery from the paronychia the thumb was wasted; it is not known if the atrophy has progressed. Cuts of the right hand have healed slowly, and corns and blisters appeared from time to time. During the last six weeks has suffered from headache on and off; otherwise has felt well, and has been steadily employed as a waitress in a restaurant.

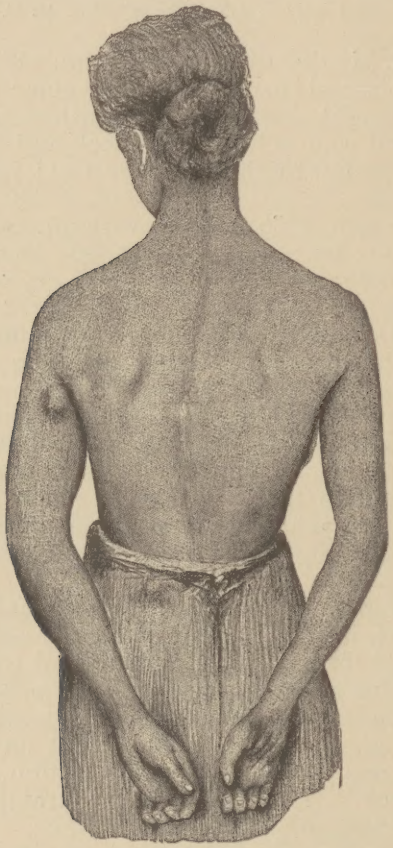
Status præsens.—A slim woman of small stature, expression bright, intelligent, but old for her age. Face symmetrical except for a possible flattening of the right cheek, teeth lost on this side. Motions of facial muscles both from volition and emotions symmetrical. Motions of eyes normal. Left pupil a trace larger than right under all conditions. Pupils react to light, accommodation, and irritation of skin of neck. Tongue projected straight, steady, no fibrillary twitching, of healthy appearance.

Lateral curvature of spine to the right in neck and upper dorsal region; right breast hangs considerably below the left, otherwise the thorax appears normal. Physical examination of heart and lungs showed nothing abnormal.

Right arm smaller than left, which careful examination shows to be perfectly normal. Complete atrophy of the

[1] Presented to the American Neurological Association Annual Meeting in Philadelphia, June, 1890.

flexor muscles of the ulnar side of the right forearm, the periform bone is freely movable. Left forearm normal. Right hand in position of main *en griffe* with complete atrophy of all the muscles except one head of the flexor brevis pollicis and a few fibres in the adductor pollicis; two corns on the palm. In spite of the extensive paralysis, the fin-



From a photograph of M. C., showing spinal curvature and atrophy of right arm.

gers are quite strongly moved and the hand efficient, as shown by her occupation. Left hand normal except for a curious hollow in the ulnar border just proximal to the base of the little finger. I do not regard this as pathological. Abduction of digit is strong and the same appearance is

occasionally to be found in healthy people. Fibrillary twitching of all the muscles of the right arm, none anywhere else.

MEASUREMENTS IN CENTIMETRES.

	L.	*R.*
Arms at axilla,	22.2	20.3
" " middle,	21.6	19.7
" " above elbow,	19.7	18.4
Forearm,	20.0	15.8
Wrist,	13.3	12.0
Knuckles,	17.1	16.5

Reaction to faradism tested with a Barrett battery, two cells; the figures being the number of millimetres which the fine secondary coil overlapped the primary. Stintsing's test electrode. Erb's largest on back of neck.

Supinator longus of right arm,	17;	left, 12.
Biceps of right arm,	20;	left, 20.

Reaction to galvanism gave K.S.Z. > A.S.Z. in left arm; and A.S.Z.> K.S.Z. in right arm, as tested with Erb's electrodes the indifferent on back part of neck, and a Hirschmann galvanometer. This galvanometer on switching the current back and forth, the circuit remaining the same, showed no changes in the strength of the current. The test was made by slowly reducing the current and reversing until K.S.Z.or A.S.Z. failed to occur. The muscles in the two arms were compared one by one.

Three weeks later tested with an Edlemann galvanometer and Stintsing test electrode, no degeneration was found, but as follows:

Right supinator longus,	K.S.Z.	1.8 m.a.	A.S.Z. 2.6.
Left " "	K.S.Z.	1.7	A.S.Z. 3.5.
Right biceps,	K.S.Z.	1.1	A.S.Z. 3.5.
Right stena muscles,	K.S.Z.	4.5	A.S.Z. 7.
Left " "	K.S.Z.	3.	A.S.Z. 8.

Also K.S.Z. was found greater than A.S.Z. with the same method and apparatus used in the first test. Therefore, either there was some error in my first test, or the reactions changed. The switching of the poles was too frequent and irregular to allow of a uniform error in reading the poles. The same frequent switching would seem to exclude any regular increase of the positive pole by return current in the right arm.

No increase of mechanical excitability of muscles; no triceps or biceps tendon reflex procured on either side.

Organs of abdomen normal.

Left knee-jerk > right; achilles tendon reflex slight, same on both sides; no ankle-clonus; no atrophy of muscles.

Right arm, quarter of body, side of neck and left leg feel "numb;" shown in figure by stippled area. The sensation seemed to be one of discomfort or consciousness rather than of true numbness.

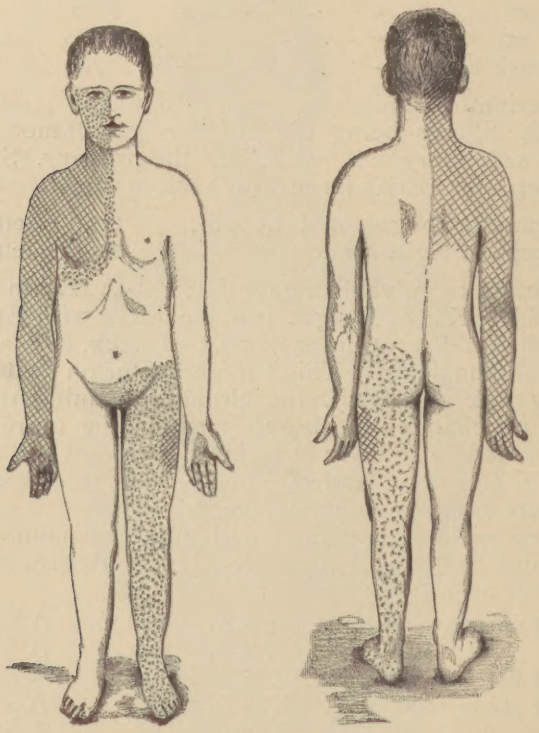


Diagram showing areas of impaired senses of heat, cold and pain (lined), and discomfort (stippled).

Sense of touch perfect all over body, not delayed. Fingers, head and point of pin and wisp of paper used in testing. Sense of temperature, as determined by the use of dry test-tubes filled with hot and cold water, diminished in an area, including the whole right side of the body from the line of the lower jaw to the seventh rib. Line of demarkation from the left side close to the middle line, distinct, but not an actual straight line. All over this area cold

(45° F.) is recognized about 70 per cent. of the trials, the percentage of error being highest an the forearms. No idea can be formed from short contact, some thirty seconds to a minute being required. A correct decision given after half a minute may be reversed at the end of a minute. With heat (130° F.) the percentage of error is higher, nearly 50 per cent. Like cold, the impairment is most on the forearm. No differences between the surfaces. The finger-tips are less affected than the rest of the limb. Patient says there is a small patch on the outside of the left thigh. In all other parts of the body replies are instantaneous, always right even with a difference of a few degrees.

The sense of pain is diminished over much the same area as the sense of temperature, but apparently varies more or less from day to day. The diminution is most marked in the right forearm. The patch in the leg is not very marked. Tests were made with the faradic wire brush, deep pinching between the fingers and working skin between the nails. Over the rest of the body there is certainly no analgesia.

Sense of position of parts perfect, as shown by recognition of slight, slow, passive motions of relaxed parts.

Location of all cutaneous irritations perfect. With eyes closed weight of objects held either one in each hand or one after the other, in either hand, very accurately determined. With faradism, painful cramp in muscles at same strength on the two sides. Sensation of mouth normal. Sight, hearing. taste, smell, good. No ataxia, no tremor, no Romberg symptom, no nerve tenderness.

Thanks to the kindness of Dr. S. G. Webber, I am enabled to report a case of syringomyelia, with autopsy, which occurred in the Boston City Hospital in 1882. As will be seen, but very few symptoms referable to the spinal cord existed, though the lesions were extensive.

"November 14th, 1882. K. D., 31 years old, single, dressmaker. Had lung fever when a child. Curvature of spine since 17 years old. Last fall caught cold after slight exposure and had a sore throat. Soon after had pain in the small of the back, shooting into both hips. Pain in back lasted about a month. Since then constant pain about dorsum of right iliac bone. Of late pain along upper border of the right scapula. At first there was pain in the left hip for three weeks. Cannot walk on account of pain above right hip, but thinks legs are as strong as ever. Limbs and

body much emaciated. No cramps or twitching; no peculiar sensations in limbs.

"No cough, dyspnœa or palpitation. Appetite poor; gaseous eructions; frequent vomiting, usually about two hours after eating; no pain in abdomen. Bowels kept regular by medicine. Micturition infrequent. Frequent sweating. T. 98.3; P. 100.

Nov. 15th.—Pain and tenderness on pressure at right sacro-iliac synchondrosis. Less tenderness over upper part of scapula. Much pain on pressure on the left side of abdomen, seems to start the pain on the right more than hurt at point of pressure.

Nov. 22d.—Nose bleed during night and continuous, less this morning.

Nov. 23d.—Nose bleed stopped yesterday morning. Now has tenderness over the sternum, which seems concentrated on the right near the junction of the fourth and fifth ribs. No fluctuation or sweating.

Nov. 29th.—Nose bleed renewed this morning.

Nov. 30th.—Only slight epistaxis about 6 A. M., on coughing.

Dec. 1st.—Slept badly.

Dec. 2d.—No abnormal sound in heart or lungs.

Dec. 7th.—Still complains of great pain in chest.

Dec. 10th.—Considerable pain this morning. Some œdema of feet.

Dec. 11th.—Nausea and vomiting.

Dec. 12th.—Urine acid, fawn-colored, ladened with urates. Sp. gr. 1024. No albumen sediment (after dissolving urates), uric acid crystals and normal epithelium.

Dec. 14th.—Has considerable pain in chest.

Dec. 15th.—Left leg numb, cannot move it well, but makes all the motions.

Dec. 16th.—Has some shortness of breath.

Dec. 19th.—Died at 5.30 P. M.

The pathologist's diagnosis ran as follows: "Chronic thickening of dura. Chronic internal hæmorrhagic-pachymeningitis. Anæmia of brain. Apparent dilatation of central canal of cord. Lateral curvature of spine. Chronic parenchymatous myocarditis. Double hydro thorax. Oedema of lungs. Chronic passive congestion of spleen. Tumor of ovary. Cancer of stomach and neighboring lymph glands."

The only parts of the pathologist's full report of interest are: "Marked curvature to right, little or no compensatory curvature in lumbar region." "Cord showed throughout its whole length a central opening varying in size from two to four millimetres." "Bodies of vertebræ occupying the seat of most marked curvature showed a soft, reddish-gray appearance on section."

I add the result of my microscopic examination of the sections lent me by Drs. Webber and Knapp.

The spinal cord was evidently not well hardened, consequently the sections are thick, and in places there is considerable shrinkage about the vessels. The sections are all stained with carmine, except one stained with hæmatoxylin. Unfortunately the sections are not marked as to level, but only by an arbitrary series of numbers from above down, nor is there any way of distinguishing the right from the left side. Therefore my statement of levels is only an inference from the structure of the sections. One side of the cord can be distinguished from the other by the continuity of the lesions, therefore, to facilitate description, one side is *arbitrarily* called right, the other left.

All the sections from the upper cervical to the lower lumbar region show changes in contour. In the upper sections the cord is flat and wide (14 x 8 mm.), in the lower part this is not so marked. The anterior fissure is wide, gaping, and the anterior columns do not bear their proper relations to each other, but are as it were partly unrolled. In the mid-dorsal region, the left half of the cord is misshapen and smaller than the right. From the mid-dorsal down the cord is of nearly normal form.

All the sections from the highest cervical to the mid-dorsal show a cavity, often large, occupying a space between the gray commissure and the posterior white commissure[2] in the cervical region, the left horn in the dorsal region. There then comes a short interval with no cavity. Lastly, a second cavity begins just to the right and behind

[2] By this I mean the small bundle of white fibre which run just in front of the columns of Goll from one posterior horn to the other.

the central canal into which it shortly breaks. This cavity is smaller than the first, and fades into the normal or slightly dilated central canal before the lowest part of the lumbar swelling is reached.

In patches throughout the cord the supporting tissue is increased. Of this no mention is made in the detailed description given below.

No. 1.—From cervical enlargement, 15 x 8 mm. shows a cavity 4.17 mm. x .81 mm., bounded in front by the gray commissure, behind by the posterior white commissure, and encroaching at both ends on the gray matter so as to nearly separate the anterior from the posterior horns. Right end of cavity slightly enlarged; left extended as a narrow cleft down middle of posterior horn. Walls of cavity formed by a mass, varying in thickness from .08 to .45 mm., of glia cells. On the outside the cells are closely packed together, have few or no processes and are round. On the inside the cells have small bodies (stained red), and many long, fine processes—that is, have the form of highly developed glia cells. The cells between are in all stages of transition. In places patches of glia cells, some of which have not stained and appear as if softened project into the lumen of the cavity. The same sort of gliomatous growth extends along the fissure in the posterior horn, the processes in most places bridging it over, thus producing a fine lace work.

Central canal filled with a mass of round cells. About forty ganglion cells in each horn; it is clear, however, that most of the group situated at the base, of the horn, have been destroyed.

A small patch in the apices of the columns of Goll, chiefly the left, is infiltrated with glia tissue, stained deep red; a few nerve-sheaths remain, but show no axis-cylinders.

No. 2.—Cervical enlargement 15. x 8. mm. The cavity is reduced to 1.05 x .81 mm, and occupies the central portion of the cord. The walls are of the same nature.

No. 5.—Upper dorsal; 10 by 7 mm. Cavity triangular with apex in left posterior horn, and base well in front, so

as nearly to destroy the gray matter. The left horns are distinctly smaller than the right, as if collapsed, and only contain six motor-cells as opposed to over thirty in the other side. Same changes in the columns of Goll, especially the right, as occurred in the first level.

No. 8.—Mid-dorsal, 10 x 7, irregular in shape. Same triangular cavity in left side, but farther back, so as to spare most of the anterior horn. Central canal still occluded.

No. 9.—Lower dorsal, 10 x 7 mm. The section is torn about the central canal, but shows no cavity.

No. 10.—Just above the lumbar enlargement, 9 x 8 mm. A long, narrow split (1.8 x .13) in the posterior horn. Walls as in the upper cavity. Central canal open, with a column cell lining.

No. 11.—Apparently same level as last, (9. x 8. mm.) Cavity in gray commissure breaking into central canal from the right and behind.

No. 13.—Cavity enlarged to 2.34 x .78 mm., involving central canal and adjacent parts. All the commissures preserved.

No. 15.—Lumbar enlargement (10. x 9. mm.). Cavity lozenge-shaped, .78 x .52, at site of central canal, in parts lined with epithelium; apparently nothing more than an enlarged central canal. No gliosis.

No. 16.—Lumbar enlargement, (11. x 9. mm.) Cavity slightly larger, of same nature. No gliosis.

It is evident that we have here a case of syringomyelia, with a surrounding gliomatous growth. The case, in itself, does not seem to me to show whether the hole is the result of the gliosis, or the reverse, but judging from other cases the first conclusion is the most probable. Indeed, if the hole is not the result of the gliosis, no shadow of a cause is apparent. The cavity is not an enlarged central canal since that structure exists in front. The nature of the lower cavity is not so clear; the lower part shows nothing definite except an enlarged central canal, but in the upper part there is undoubted gliomatous growth and a cavity separate from the canal.

A point of interest is the extensive destruction of the gray matter, especially of one posterior horn, but also in places (upper cervical and mid-dorsal), extensively involving the anterior horns. It is clear that for a considerable distance in the dorsal cord the majority of the ganglion cells were destroyed. The same is true of the cells at the base of the anterior horns in the cervical region. The cavity in the other parts gives one the impression of having expanded and driven the tissue before it rather than to have infiltrated and destroyed the tissue. It is worthy of note that the commissures between the anterior columns, the gray commissure and the narrow band of white substance posterior to the gray commissure are preserved, as well as a connection between the two horns of the same side. This may explain in part the preservation of functions, but how about the lost ganglion-cells?

The degenerated spots in the column of Goll are small, and do not seem to be of so much importance.

The few symptoms which can be referred to the cord seem remarkable when compared with the extensive lesions found, and would imply carelessness on the part of the house officer in writing the records, were it not that many similar cases are on record. Dr. Webber also informs me that the cord disease was suspected, and the patient most carefully examined in all respects, except for senses of heat and cold.

The infrequent micturition and some of the pains may have sprung from the cord, but the other troubles would seem to sufficiently explain them. The numbness of the left leg was probably of central origin. I am inclined to believe that the spinal curvature was of trophic origin. It could not have been the result of cancer of fourteen years duration, nor even tubercular. In either case, a more definite report from the pathologist might fairly be expected.

The first case shows a perfect history of one of the groups of symptoms caused by syringomyelia, and with our present knowledge, must be accepted as such. Since it is only of late years that the disease has received consideration, it may not be out of place to review the subject before passing on to the subject of diagnosis.

Syringomyelia means a hole in the spinal cord extending as a tube up and down for a greater or less extent. This condition very likely results from a number of causes, but seems, as claimed by Schultze,[3] chiefly to depend upon the absorption of a more or less dense gliomatous tumor, having a marked tendency to invade only the gray matter and the apices of the posterior columns. The upper part of the cord is also more affected than the lower part. On this peculiarity of location depends our power to diognosticate the disease. The axis-cylinders in the affected area are quickly destroyed, do not remain as in multiple sclerosis.

Somewhat the same sort of cavities, but more extensive, have been found in the cord in cases of sarcoma (Harris[4]). There may also be cases due to myelitis and softening, (*e. g.* Oppenheim,[5] Silcock,[6] Van Giesen[7]); cases due to hæmorrhage; cases resulting from imperfect junction of the cord along its posterior border. Cases of enlargement of the central canal, true hydromyelia, do not concern us clinically. I make no positive statement, as the whole subject is still under debate.

Owing to the peculiar localization of the disease, a definite group of symptoms is produced which renders a diagnosis possible in quite a proportion of cases. The onset of symptoms may be gradual, without cause, or may develop suddenly after a severe fall or the like. The cardinal symptoms are muscular atrophy, loss or impairment of senses of temperature and pain, the other senses being normal, and trophic disturbances varying from bullæ to necrosis of phalanges, deep cellulitis and the like. The upper part of the body is chiefly affected. The legs are apt, owing to descending degeneration, to become spastic-paretic. In the arms the atrophy is frequently of the Aran-Duchenne type, destroying all the small muscles of the hand, and then

[3] Schultze, Zeitschrf. f. Klin. Med., No. 13, p. 523, 1887; Virchow's Archiv vol. 102, p. 435, 1885; Ibid., vol. 87, 1882.

[4] Harris, Brain, vol. viii., p. 447, 1885.

[5] Oppenheim, Charité Annalen, xi., p. 409, 1886.

[6] Silcock, Trans. Path. Soc., London, 1887–88, p. 18.

[7] Van Giesen, Journal of Nervous and Mental Disease, 1889, p. 393.

working up by regions, not by nerves. Main *en griffe* appears, but the hand is much more useful than might be expected. Fibrillary twitching may or may not exist, when atrophy is complete no reaction occurs. Where fibres are left K.S.Z. may be greater or less than A.S.Z. About this time or before, patient rarely knows when, the senses of pain and temperature are impaired or lost. The change may be one of simple diminution only, differences of five or ten degrees being noted; or complete, severe injuries from burns and scalds then resulting. Sometimes the sense for higher temperature is lost, say from 35° C. up, while that for lower temperature is retained fairly well. The analgesia may be extreme, such that necrosed phalanges can be excised without pain. A hand, arm, quarter of the body or more may be affected, the area of loss of pain and temperature senses roughly coinciding.

It is worthy of note that the areas of impaired sensibility follow the distribution of such troubles in hysteria, that is, involve a hand, forearm or arm, not the radial or ulnar border of the arm as in trauma of the cord.

The sense of discomfort in the affected limbs, as described in case I., may be due to the brains missing the sensations of heat, cold and pain, the equivalent of "vision obscure" of the French.

The impaired sensibility leads to the infliction of many injuries and their subsequent neglect, which is probably responsible for many of the "trophic" changes. But there are apt to be œdema, formation of bullæ and the like, of undoubted nervous origin. In most cases more active inflammatory troubles occur, as loss of nails, necrosis of phalanges, deep inflammation in the arms, necrosis of arm bones or similar processes in the feet. Sometimes active inflammatory troubles form the leading symptom, the atrophy is slight, the impaired senses not noted.

Spontaneous painless fractures of the bones of the upper limbs is also a peculiar symptom, at times the only conspicuous one. Patient, usually a powerful man, comes to the hospital with a broken ulna, radius or metacarpal, with a history of having suddenly lost control of the mem-

ber some days before. It is the swelling or uselessness which brings him in, not pain for there is none. Such fractures are apt to do well—knit as in healthy people. In some cases there may be a trophic change in the bones, but in Schultze's case with autopsy, there certainly was not. These fractures seem to be due to the unconscious use of extreme force, a point of some interest, as they occur in cases where senses of touch and position are normal; sense of resistance not noted.

The legs may show precisely the same symptoms, with or without the arms being affected. But owing to descending degeneration from the more advanced changes in the upper part of the cord, are apt to be more or less in a state of spastic paresis. At times the two classes of changes are blended, producing a confusing symptom complex.

A prodromal symptom not rarely noted by the patient and present in many cases, is that of pain in the spine between the shoulders and radiating over the body on sneezing. Neuralgic pains often occur, located chiefly in the affected regions. The pains may be furious, all sorts of paræsthesia are frequently present and may go far toward making life miserable. Such are the symptoms which are of value for diagnosis, many others are liable to occur, almost any which can result either from irritation or destruction of parts of the cord. Thus sometimes early, frequently before the end, the sense of touch is seriously impaired, also the sense of position, resulting in total anæsthesia, or hyperalgesia may exist.

The head is not much affected unless some secondary or complicating trouble, as cerebral gliomia supervenes. Through the sympathetic the pupils may be large and sluggish, the lid droop and the eye be sunken. Towards the end true bulbar symptoms may supervene and carry off the patient,

The bladder, rectum, escape till late, if affected at all.

Death results from general wasting, bed-sores and the like, as in other spinal troubles, from bulbar symptoms, or from septic infection resulting from the inflammatory troubles of the affected members.

It must not be forgotten that the above description is only intended for that class of cases which run a typical course, the group which is recognizable. There are a good many cases showing the symptoms of multiple tumor, myelitic fossæ and the like, which are not recognizable; also cases as with cerebral tumor occur without any symptoms. Of this case II. is a marked example.

DIAGNOSIS.

Where the three cardinal symptoms of atrophy, loss of senses of heat, cold and pain exist, the difficulties of diagnosis, barring a few diseases, is slight.

The diseases which appear to need special consideration are pareso-analgesia of Morvan, hysteria, leprosy, neuritis, progressive muscular atrophy and small focal lesions in the posterior part of the internal capsule.

Morvan,[8] from a small district in France with a population of 50,000, has described a group of some seventeen cases which closely resemble cases of syringomyelia. Schultze and others have pronounced them to be cases of syringomyelia; this Morvan has steadily denied. According to Morvan a distinction is to be made by: 1. The much more marked trophic troubles and arthropathies. 2. The constant impairment of the sense of touch.

A study of the history of his cases certainly gives a different impression from that of a similar number of cases of syringomyelia. The muscular atrophy seems to be less pronounced, the legs are comparatively exempt, and the trophic troubles distinctly more pronounced. Not a few of the descriptions might be taken for partial descriptions of leprosy.

While it is too early to make any positive statements in regard to pareso-analgesia, Morvan's position is certainly supported by an autopsy made by Gambault, in which a peculiar thickening of the nerves, a sclerosis of the cortical zone of the cervical cord, and thickening of the arteries

[8] Morvan, Gazette hebdom. de Med. et Chir. Aug. 30th, Sept. 6th, 1889; Ibid No. 32, et seg., 1886.

was found. In another case the nerve of an amputated finger showed the same sort of changes.

To my mind, Morvan's position is also strengthened by the large number of cases occurring in a small population, while syringomyelia is a rare disease of general distribution. It certainly looks as if *Mal de Morvan* might be hereditary or due to some habit, local custom or occupation, but time and autopsies alone can settle the question.

As Charcot[9] has lately pointed out hysteria can produce a symptom complex closely resembling syringomyelia, as special paralysis of the senses of pain and temperature, paralysis, trophic disturbance and atrophy. One or two of the reported cases of syringomyelia certainly read very much like hysteria. Hysteria should, however, be distinguished by careful observation, history and course. When either disease is at all well marked there can be no doubt.

In my case, as in many others, it might be held that the patient really suffered from two diseases, progressive muscular atrophy and hysteria. But there is none of the hysterical mien or other symptoms of hysteria, while the whole can be explained by one cause.

Anæsthetic leprosy offers considerable difficulties of diagnosis at times, but with care can probably always be excluded. Unfortunately, a clear conception of leprosy is rendered difficult by the inclusion of a certain number of cases of syringomyelia, as the cases of Langham[10] and Steudener.[11] According to Leloir,[12] upon whose work I have chiefly drawn, the anæsthesia of leprosy has two origins. In one class of cases it is due to the injury of the terminal nerves in the skin by infiltration, the same as in lupus and other skin diseases. In these cases there is or has been a visible cutaneous lesion, and the affected areas occur in patches. The anæsthesia may be total, be general, but nowhere complete, or affect only sense of pain or temperature. These cases should offer no difficulty of diagnosis,

[9] Charcot, Bull. Med., Paris, iii., 787, 1889.

[10] Langham, Virch. Archiv., vol. lxiv., p. 475, 1875.

[11] Steudener, Beitrage zur Pathologie der Lepra mutilans, Erlangen, 1867.

[12] Leloir, Praite practique et theorique de la Lepre, Paris, 1886.

especially as a piece of skin can be punched out and examined for the lepra-bacillus, which is usually abundant in the skin lesions. In the other class of cases, the pure lepra nervorum, there is muscular atrophy and partial sensory paralysis due to a leprous thickening in the nerves. At times, touch is normal, senses of pain and temperature being the same as in syringomyelia. Judging by Leloir, the mass of these cases show at one time or another skin lesions which are sufficient to separate them from syringomyelia, but all acknowledge that at times there may be no surface manifestations. None of the authors known to me, however, give clear histories of such cases. Neither the case of Jacobi or Morrow can be so classed.

Granted that such a case, or one in which the skin symptoms have passed off and are denied, there still remain several points of value.

The atrophy and partial sensory disturbance is limited to certain nerves, not segments. Even when all the nerves, say of the hand, are affected, the border is irregular, and judging by decriptions, the sensory paralysis fades away. In leprosy the nerves are thickened, in syringomyelia this is not the case, unless deep inflammation has existed. In syringomyelia, on the other hand, there are apt to be a host of other symptoms pointing to central trouble, as signs of secondary degeneration up or down.

Though Jacobi[13] enlarges upon the great difficulty of diagnosis he arrives at one which it would be difficult to question. With Morrow's[14] case the conditions are different it would seem, judging by the skin eruption, contractures, loss of all senses, irregular border, to be a case of leprosy, certainly these characters are not special signs of syringomyelia. Unfortunately the description of the case is more or less contradictory with itself and the figure, and also leaves important points in doubt. Progressive muscular atrophy is excluded by the absence of loss of the surface senses.

[13] Jacobi. Jour. of Nervous and Mental Diseases, No. 6, 1886.

[14] Morrow. Jour. of Cutaneous and Genito-Urinary Diseases, vol. viii., p. 1, 1890.

Multiple neuritis is said at times to produce more or less similar conditions to those of syringomyelia, but in the great majority of cases there is no partial sensory paralysis. In all cases the disturbance is by nerve areas. Picked sensory paralysis, due to diseases of the nerves, are rare, and but few cases of loss of temperature sense or analgesia are on record in which the pathology is known. The cases of Jacobi and Ziehl[15] depended upon local injury and infiltration of the nerve. Pick's[16] case depended upon exposure to cold, and is by no means clear as to the seat of trouble. The report of Berger's[17] case gives no evidence as to pathology, and by no means excludes hysteria.

Cerebral trouble may produce partial sensory paralysis as well as muscular paralysis. In these cases there is no such atrophy as in syringomyelia, and the paralysis is of the monophlegic or hemiphlegic type.

It is hardly necessary to say that a large number of cases of syringomyelia vary so much from the type as to acquire a special line of reasoning for their diagnosis, provided they do not baffle all efforts.

To those who desire to study up the literature the summary of 116 cases by Baumler,[18] and a recent review by Buhl,[19] will give the requisite start. The American articles are by Starr,[20] Upson,[21] Van Giesen and Booth.[22]

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[15] Ziehl. Deut. med. Woch., No. 17, p. 335, 1889.
[16] Pick. Wien. med. Woch, p. 617, 1889.
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